REMARKS

This paper is being filed in response to the Office Action, dated March 23, 2004, that was issued in connection with the above-identified application. Applicants respectfully request entry and consideration of the amendments and remarks presented herein.

Claims 1-11 are pending in the instant application. Claims 1-2, 4-6, and 8 have been cancelled herein without prejudice. Claims 3 and 9 have been amended herein. The amendments to claims 3 and 9 are supported by the specification as initially filed, for example, at paragraphs [0010] and [0014] and, therefore, do not constitute new matter.

Claims Are Patentable Over Beattie

Claims 3, 7, and 9-11 have been rejected under 35 U.S.C. § 102(b) as allegedly anticipated by Beattie et al., 1996, *Virus Genes* 12(1):89-94 (hereinafter "Beattie"). The Examiner has alleged that Beattie teaches expression vectors comprising vaccinia virus devoid of the E3L gene and exogenous DNA encoding a reovirus σ3 protein antigen operably linked to an expression control sequence. The Examiner has alleged that Beattie teaches the claimed deletion due to the recitation of the open term "having."

Applicants traverse this rejection and assert that Beattie fails to teach each and every element of the claimed invention. Claims 3, 7, and 9-11, as amended herein, relate to a vaccinia virus vector comprising a nucleic acid sequence that encodes an E3L protein that lacks native amino acids 1-54 and is capable of binding double stranded RNA. Beattie, by contrast, teaches a vaccinia virus vector that does not encode **any** of the amino acids of the E3L gene. *See* Beattie, page 90, col. 2, first paragraph *citing* Beattie et al., 1995, *J. Virol.* 69(1):499-505 (made

of record by Applicant's IDS filed August 1, 2001 and considered by the Examiner on March 14, 2004). Thus, Beattie necessarily fails to teach a recombinant vector encoding an E3L gene product of any kind, much less one that binds dsRNA.

Moreover, vectors of the present invention that encode an E3L protein that lacks the amino-terminal 54 amino acids maintain viral replication, protein synthesis, and interferon-resistance that is indistinguishable from wild-type virus. *See e.g.* Application, paragraph [0021]. By contrast, deletion of the entire E3L gene results in vectors that lack wild-type replicative capacity, and interferon resistance. *See* Beattie, page 90, col. 2, second paragraph (replicative capacity compromised in all cell types tested except chick embryo fibroblasts). Therefore, since Beattie clearly fails to teach each and every limitation of claims 3, 7, and 9-11, Applicants respectfully request withdrawal of this rejection.

Applicants believe that the claims are in condition for allowance and, accordingly, respectfully request prompt, favorable action.

Although Applicants do not believe any fee is presently due, the Commissioner is hereby authorized to deduct any fees required with this submission not otherwise enclosed herewith from Deposit Account No. 02-4377. Two copies of this paper are enclosed.

Respectfully submitted,

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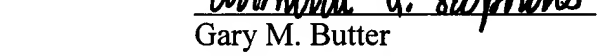

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